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Differential effects of stimulus characteristics during knee joint perturbation on hamstring and quadriceps reflex responses

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When stretching muscles of the ankle joint, stretch velocity and amplitude were shown to selectively influence specific parts of the stretch reflex. The present study investigated whether similar effects can be observed at the knee joint. Seventeen subjects were exposed to sudden anterior tibial translations. The influence of stimulus amplitude was analyzed by applying a low (LIMP) or high impulse (HIMP). To investigate the influence of velocity, rate of force development of the perturbation was chosen either low (LRFD) or high (HRFD). Activation of biceps femoris (BF), semitendinosus (ST), vastus medialis (VM) and vastus lateralis (VL) was calculated in four consecutive timeframes (P0, P1, P2, P3). During P1, RFD (ST: $p < .05$; BF: $p < .01$; VM: $p < .05$; VL: $p > .05$) and during P2, impulse (ST: $p < .05$; BF: $p < .01$; VM: $p < .01$; VL: $p < .01$) did significantly influence reflex activation. The present study showed that stimulus characteristics influenced specific reflex components of knee joint muscles. As only hamstring muscles were stretched, whereas quadriceps was unloaded, it is concluded that different mechanisms like homonymous and heteronymous muscle afferents as well as joint and skin afferents might contribute to the reflex responses.

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1. Introduction

Anterior cruciate ligament (ACL) rupture is one of the most common injuries in sports performance and produces long recovery times which are associated by high socio-economical costs. An estimated amount of 50 million Euro are spent for treatment after ACL ruptures in Germany each year (Beck, Reichelt, & Beck, 2002; Nebelung, 2006; Schabus & Bosina, 2007). Thus, it is of utmost importance to understand the genesis of this type of injury and, consequently, the protective mechanisms which may prevent it. Stress to the ACL is induced when the tibia is anteriorly translated relative to the femur. This anterior tibia translation is counteracted by the activation of the hamstring muscles. In this context, several previous studies have focused on reflex responses of the hamstrings as a protective mechanism following a sudden tibia translation. Indeed, evidence has been provided that there is a relationship between the magnitude of neuromuscular reflex responses and the stiffness of the knee joint (Dhaher, Tsoumanis, Houle, & Rymer, 2005; Jennings & Seedhom, 1994; Olmstead, Wevers, Bryant, & Gouw, 1986; Wojtys & Huston, 1994). However, in most studies investigating stretch reflexes of muscles controlling the knee joint, the fundamental properties of translational perturbations, namely the amplitude and the velocity, were not addressed. For the ankle joint, it was shown that latency and magnitude of reflex responses are determined by the stretch velocity and the amplitude. For instance, the short latency response (SLR) of the soleus muscle which is considered to be mediated by fast conducting Ia afferent fibers (Bove, Nardone, & Schieppati, 2003; Morin & Pierrot-Deseilligny, 1977), was reported to be sensitive to the stimulus velocity (Gollhofer & Rapp, 1993; Grey, Ladouceur, Andersen, Nielsen, & Sinkjaer, 2001; Leukel et al., 2009). The size of the medium latency response (MLR), on the other hand, was determined by the amplitude of the induced stretch (Gollhofer & Rapp, 1993; Leukel et al., 2009). The MLR has been attributed to oligosynaptic excitation of spinal motoneurons via group II afferents (Bove et al., 2003; Grey et al., 2001; Nardone & Schieppati, 1998; Nardone & Schieppati, 2004) and possibly by group Ib afferents (Dietz & Duysens, 2000).

The aim of the present study was to induce knee joint perturbations with different velocities and amplitudes to investigate functional reflex responses in quadriceps and hamstring muscles. Thereby, it was hypothesized that the stretch velocity and the amplitude of the stretch affect different components of the stretch reflex response analogue to the findings of muscles controlling the ankle joint. The velocity of the externally applied mechanical (anterior tibial) perturbation was determined by the rate at which the perturbing force developed, whereas the magnitude of the perturbing impulse that was transferred predefined the stimulus amplitude. Either the stimulus velocity or the amplitude during perturbation was altered in order to investigate the effect on electromyographic responses of knee flexors (hamstring muscles) and extensors (quadriceps muscles).

2. Methods

2.1. Participants

Seventeen male subjects (age: 23.4 ± 1.9 years; height: 180.0 ± 6.1 cm; weight: 76.9 ± 7.1 kg; *M* ± *SD*) with no history of orthopaedic or neurological disorders volunteered to participate in the study. All subjects gave written informed consent before the experiment. The study was approved by the local ethical committee and all experiments were carried out according to the Declaration of Helsinki.

3. Experimental procedure

The subjects were standing in full weight-bearing bipedal stance with the arms held at the waist with a knee flexion angle of 30°. The knee flexion position enabled a translation of the tibia in relation to the femur into anterior direction (Markolf, Graff-Radford, & Amstutz, 1978; O'connor, 1993). Testing was performed on the right leg. To avoid anticipation of the stimulus, subjects were visually and acoustically uncoupled from the perturbation proceeding.

Dynamic tibial translation in posterior–anterior direction was induced using a rope and pulley system. The rope was connected to a circular band-sling applied around the shank and thereby

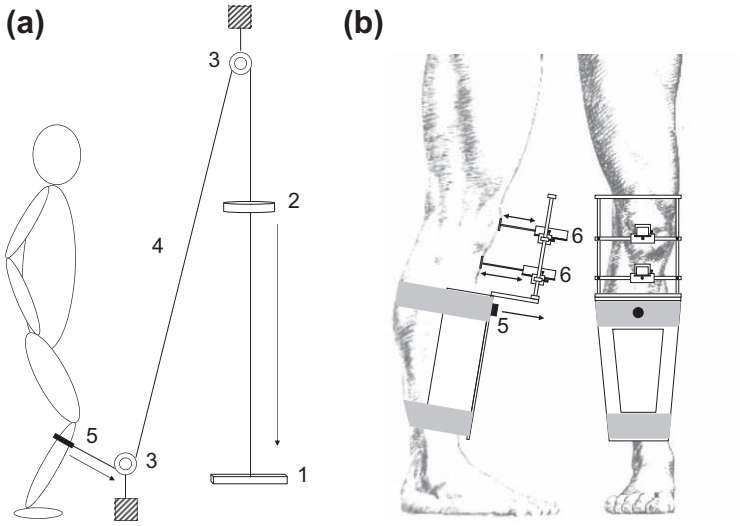

Fig. 1. A: Experimental setup, B: Measurement system 1: stopper, 2: falling barbell weight, 3: pulley, 4: rope, 5: force transducer, 6: linear potentiometer. Arrows are indicating the direction of the force.

transferred the impulse of a falling barbell-weight to the dorsal side of the lower leg. The pulling system was positioned perpendicular to the longitudinal axis of the tibia and in parallel to the tibial plateau (Fig. 1a).

To control the force applied to the shank, a force transducer (measuring range: 0–5000 N, sensitivity −3.42 to 3.36 pC N^{-1}, linearity ±0.2 to 0.3%, Kistler, Winterthur, Switzerland) was inserted between the pulling rope and the band-sling. Maximal force (Fmax) was derived from force time curves.

The mechanical perturbation can be characterized by the magnitude of the impulse (IMP), represented by the area beneath the force time curve and the rate of force development (RFD), represented by the average tangential gradient of the force time curve. In order to selectively alter amplitude or velocity of the stimulus, IMP and RFD could be modulated independently by variation of the mass of the barbell-weight and the drop height. Four conditions were tested (for exact parameters see Table 1): IMP was set to low (LIMP) or high (HIMP) while RFD was kept constant. RFD was set to low (LRFD) or high (HRFD) while IMP was kept constant. The four different conditions were applied in a randomized order to avoid systematic effects of fatigue or habituation. At least six successive trials per condition were performed. If one or more trials were unsuccessful, they were repeated. This was the case, if subjects changed their positioning or if EMG-signals were not satisfying. The decision was made on the basis of visual online control during data acquisition. The single trials were separated by breaks lasting 6–12 s. Duration of the breaks was altered randomly to avoid anticipation. In order to familiarize the subjects with the perturbation and the measurement device, ten stimuli were applied (RFD: 2.32 ± 0.25 N/ms; IMP: 4.24 ± 0.29 Ns) before the measurement was started.

3.1. Measurement of anterior tibial translation

During perturbations, knee joint stability was measured by quantifying posterior–anterior tibial translation using two linear potentiometric position transducers (measuring accuracy: <0.01 mm, linearity ±0.7%, Type CLR13-50 Megatron®, Putzbrunn, Germany). The potentiometers were positioned on a mounting frame applied to the ventral side of the shank. The mounting frame was fixed in parallel to the longitudinal axis of the tibia. One potentiometer was attached to the patella representing the position of the distal thigh. To control for movement of the mounting frame in relation to the shank, a second potentiometer was attached to the tuberositas tibiae representing the position of the proximal shank. The measuring sections of the two linear potentiometers were aligned perpendicular to

Table 1
Stimulus conditions.

Parameter		LIMP	HIMP	*p*	LRFD	HRFD	*p*
IMP (Ns)	Mean	3.18	8.32	**.000**	3.72	3.79	.642
	SD	0.56	0.27		0.23	0.50	
RFD (N/ms)	Mean	2.10	1.92	.105	1.73	2.50	**.000**
	SD	0.36	0.18		0.22	0.28	
Fmax (N)	Mean	251	403	**.000**	264	301	**.000**
	SD	22	11		7	16	
Weight (kg)		1.25	5.0		2.5	1.25	
Height (m)		1.0	0.4		0.5	1.5	

the longitudinal axis of the tibia and parallel to the tibial plateau in the sagittal plane (Fig. 1b). Positioning of the measurement device was not changed during the entire experiment. The tibial translation was calculated as the difference between the covered distances of thigh and shank against the mounting frame. Maximal amplitude and average velocity of tibial translation from onset to maximum were derived from the translation-time curve.

3.2. Neuromuscular responses

Bipolar surface electrodes (Hellige, Germany) (diameter 10 mm, center-to-center distance 25 mm) were placed over the muscle bellies of semitendinosus (ST), biceps femoris (BF), vastus medialis (VM), and vastus lateralis (VL). A reference electrode was placed on the patella. Inter-electrode resistance was kept below 2 kΩ by means of shaving, light abrasion, and disinfecting of the skin. Electrodes were directly connected to custom built preamplifiers (gain 200, input impedance 4000 MΩ, common mode rejection 75 dB at 60 Hz). The preamplified signals transmitted to the main amplifier (band-pass filter [sixth-order, 10 Hz-1 104 kHz], gain 6.25 [overall gain 1250]). Force, tibial translation, and EMGs were synchronously sampled at 2 kHz. Data collection and processing was performed using Lab View® based software (National Instruments®, Austin, Texas, USA).

3.3. Data analysis

Six successive stimuli were applied for each condition (LIMP, HIMP, LRFD, HRFD). The corresponding EMG signals were rectified and averaged for each subject and condition.

To detect the onset, mean and standard deviation (*SD*) of EMG amplitude were calculated 100 ms prior to stimulus application. A horizontal cursor was set three standard deviations above the mean of the EMG baseline amplitude. Onset of EMG was defined by the first major deflection of the EMG signal exceeding the horizontal cursor (De Luca, 1997). Reflex Latency was calculated as the difference between the onset of the tibial translation signal and the onset of the EMG signal.

The data were analyzed according to Mrachacz-Kersting, Lavoie, Andersen, and Sinkjaer (2004). The tibial translation signal has been used as the indicator for the perturbation onset. P0 was defined from the onset of the tibial translation to 50 ms before. P1 was defined from 20 to 40 ms after the onset of the tibial translation. P2 was defined from 40 to 60 ms after tibial translation started. P3 was defined from 60 to 95 ms (Fig. 2).

EMG signals were integrated for each of the timeframes (iEMG). Time constrains preclude reflex responses to occur within P0, therefore, the iEMG in this time frame was used as a reference (background activity). Consequently, the reflex induced gain was calculated as the activation during the particular timeframe (P1, P2, P3) relative to the background activity (BGA = P0). Values are reported as mean ± standard deviation (*SD*).

3.4. Statistics

Differences in IMP and RFD caused by the stimulus conditions were controlled using Student's paired samples *t*-tests (Table 1).

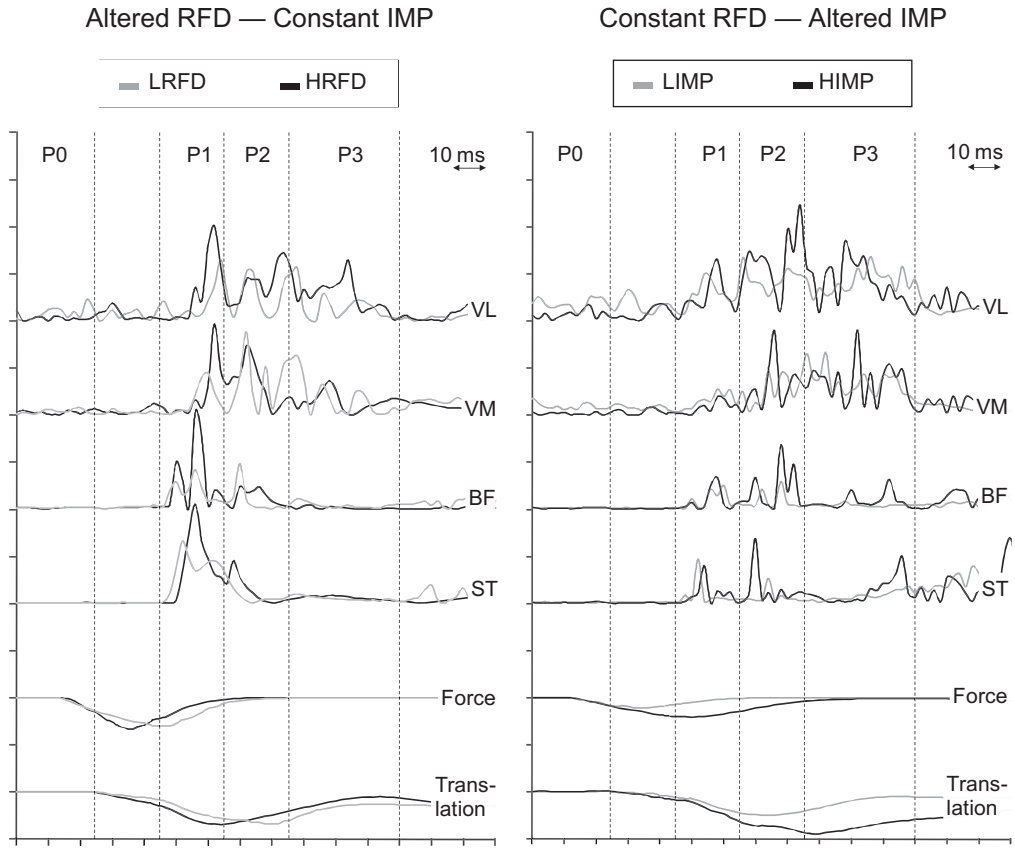

Fig. 2. Average of single subject comparing stimulus conditions LRFD vs. HRFD and LIMP vs. HIMP (ST = M. semitendinosus, BF = M. biceps femoris, VL = M. vastus lateralis, VM = M. vastus medialis). Negative values for tibial translation and force are indicating anterior direction. P0 indicating background activity was defined from the onset of tibial translation to 50 ms before. P1 was defined 20–40 ms after the onset of tibial translation. P2 was defined 40–60 ms after the onset of tibial translation. P3 was defined 60–95 ms after the onset of tibial translation.

The effects of IMP and of RFD on tibial translation (maximum, maximal velocity) and neuromuscular activation (onset latency, iEMG during P0, P1, P2, P3) were analyzed by means of Analysis of Variance (ANOVA) using post-hoc tests (Bonferroni). *P*-values less than .05 were considered statistically significant.

To assess correlation between stimulus conditions (IMP, RFD) and tibial translation (maximum, maximal velocity), Pearson's correlation was calculated. Statistical analyses were executed using SPSS (IBM SPSS Statistics Version 19, SPSS Inc., Chicago, IL).

4. Results

4.1. Effects of the stimulus characteristics on the mechanical anterior tibial translation

Maximal tibial translation differed significantly, $F(1, 32) = 46.573$, $p = .000$, when IMP was altered (LIMP: 5.3 ± 1.3 mm; HIMP: 9.2 ± 1.8 mm) while no changes, $F(1, 32) = 3.468$, $p = .072$, were observed for the velocity of tibial translation (LIMP: 0.36 ± 0.09 m/s; HIMP: 0.41 ± 0.06 m/s).

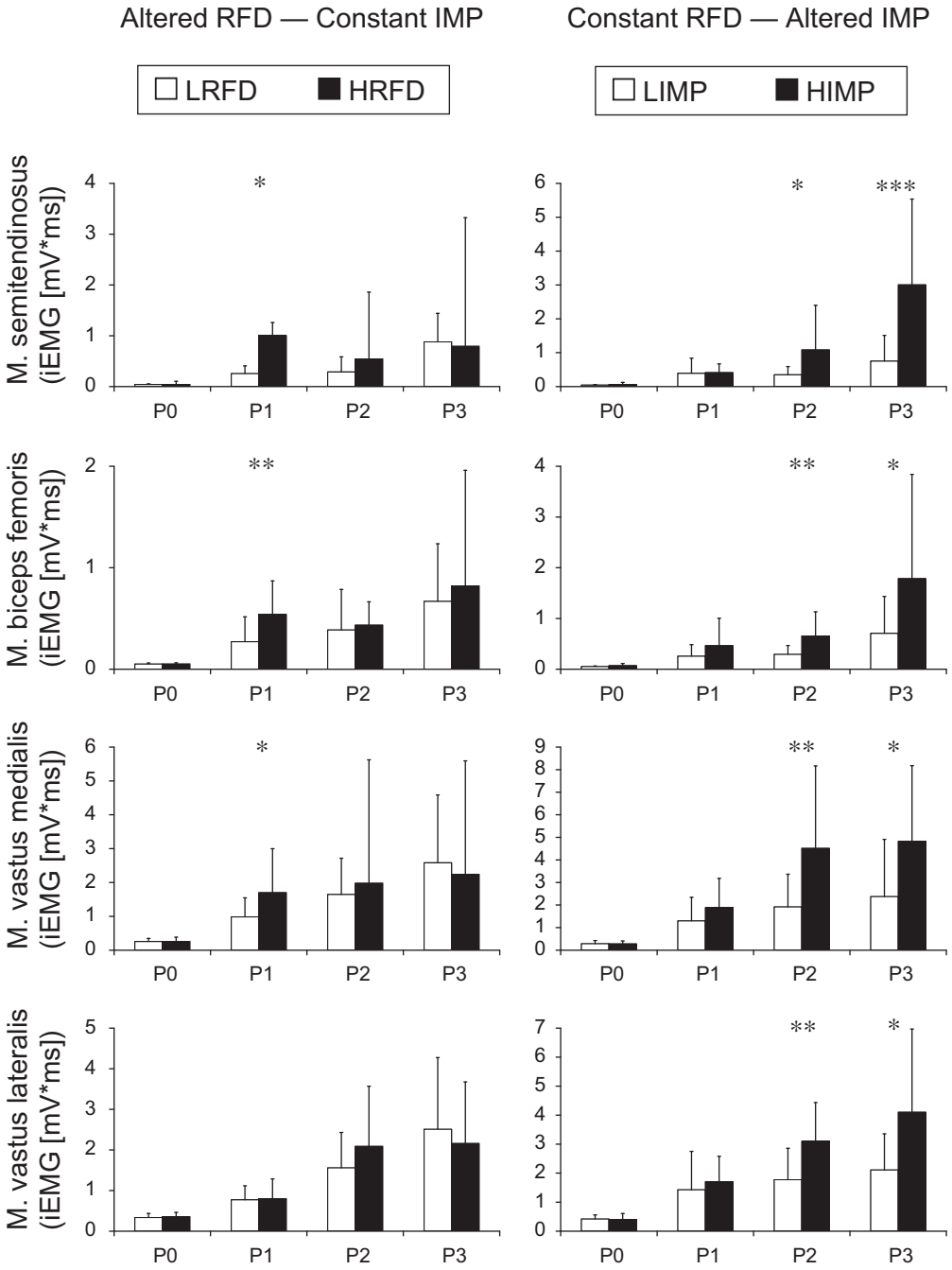

Fig. 3. Comparisons of muscle responses (iEMG) to different stimulus conditions, values are expressed as mean + SD. Different muscles are displayed in different rows. Different stimulus conditions are displayed in different columns and different bars. Asterisks are indicating statistical significance of differences in means between the conditions (*: $p < 0.05$; **: $p < 0.01$; ***: $p < 0.001$).

Table 2a
ANOVA of RFD (df = 1,32) on neuromuscular activation during the four timeframes.

Muscle	Semitendinosus		Biceps Femoris		Vastus Medialis		Vastus Lateralis	
Timeframe	*F*	*p*	*F*	*p*	*F*	*p*	*F*	*p*
P0	.067	.797	.000	.993	.008	.930	.255	.617
P1	**5.898**	**.021**	**7.415**	**.010**	**4.615**	**.039**	.043	.837
P2	2.618	.115	.188	.667	.487	.490	1.617	.213
P3	.205	.654	.246	.624	.192	.664	.385	.539

Table 2b
ANOVA of IMP (df = 1,32) on neuromuscular activation during the four timeframes.

Muscle	Semitendinosus		Biceps Femoris		Vastus Medialis		Vastus Lateralis	
Timeframe	*F*	*p*	*F*	*p*	*F*	*p*	*F*	*p*
P0	1.442	.239	3.647	.065	.022	.884	.064	.802
P1	.025	.876	2.153	.152	2.113	.156	.519	.477
P2	**5.151**	**.030**	**8.823**	**.006**	**7.466**	**.010**	**10.043**	**.003**
P3	**12.362**	**.001**	**4.185**	**.049**	**5.801**	**.022**	**6.918**	**.013**

Table 3
Stimulus induced gain of muscle activity during respective timeframe in relation to background activity (P0).

	Muscle	Semitendinosus			Biceps Femoris			Vastus Medialis			Vastus Lateralis		
Condition		P1	P2	P3	P1	P2	P3	P1	P2	P3	P1	P2	P3
Low RFD	Mean	6.0	6.7	12.1	5.0	11.1	7.1	4.0	6.9	5.9	2.5	4.9	4.5
	SD	3.7	6.5	7.6	3.9	8.6	5.5	2.2	4.5	4.7	1.4	2.7	3.0
High RFD	Mean	32.2	14.2	11.4	10.5	8.5	8.4	7.2	8.0	5.3	2.4	6.1	3.6
	SD	24.2	13.2	10.2	6.4	4.5	9.4	6.0	6.5	6.1	1.6	3.9	2.3
	F	**5.170**	2.914	.050	**10.049**	.000	.213	**4.338**	.333	.096	.043	1.060	1.063
	p	**.030**	.097	.824	**.003**	.990	.648	**.045**	.568	.759	.838	.311	.310
Low IMP	Mean	11.9	7.9	10.3	4.7	5.5	7.4	5.5	6.8	4.7	4.0	4.7	3.2
	SD	9.1	6.0	11.4	3.7	2.9	6.5	6.0	4.4	3.3	4.0	3.3	2.4
High IMP	Mean	13.5	46.7	65.8	5.7	12.2	16.8	7.0	19.1	10.8	4.9	8.5	6.9
	SD	11.8	29.4	121.1	3.1	11.9	15.3	4.4	18.1	7.6	3.3	4.5	5.4
	F	.375	**4.292**	3.382	.804	**4.378**	3.177	.724	**7.307**	**9.297**	.624	**8.725**	**6.727**
	p	.545	**.046**	.075	.376	**.044**	.084	.401	**.011**	**.005**	.435	**.006**	**.014**

ANOVA for Conditions (High vs. Low; df = 1,32).

Differences in maximal tibial translation were not significant, $F(1, 32) = 0.019$, $p = .892$, when RFD was altered (LRFD: 6.2 ± 1.2 mm; HRFD: 6.1 ± 1.3 mm), whereas translation velocity (LRFD: 0.35 ± 0.07 m/s; HRFD: 0.44 ± 0.09 m/s) was significantly altered, $F(1, 32) = 6.552$, $p = .015$, with RFD.

Maximal tibial translation correlated significantly with IMP ($r = .629$, $p = .000$) but not with RFD ($r = .000$, $p = .999$), while translation velocity correlated significantly with RFD ($r = .676$, $p = .000$) but not with IMP ($r = .077$, $p = .445$). Fmax correlated significantly with both amplitude ($r = .581$, $p = .000$) and velocity ($r = .581$, $p = .000$) of the tibial translation indicating that Fmax was no adequate parameter to discriminate particular stimulus characteristics.

4.2. Effects of the stimulus characteristics on the muscle reflex responses

Muscle onset latencies in relation to the onset of tibial displacement (ST: 22.2 ± 2.9 ms; BF 21.9 ± 1.9 ms; VM 29.7 ± 2.7 ms; VL 30.1 ± 2.8 ms) were about 14 ms shorter than when the onset of force served as a reference. No significant effect of RFD (ST: $F(1, 32) = 0.633$, $p = .431$; BF: $F(1, 32) = 0.844$, $p = .365$; VM: $F(1, 32) = 0.216$, $p = .645$; VL: $F(1, 32) = 0.422$, $p = .521$, or of IMP [ST: $F(1,$

32) = 0.012, p = .915; BF: $F(1, 32) = 0.022$, p = .884; VM: $F(1, 32) = 0.219$, p = .643; VL: $F(1, 32) = 0.481$, p = .483, on reflex latencies could be detected for any of the investigated muscles.

Stimulus dependent variations in neuromuscular activation are illustrated in Fig. 3. Corresponding results of ANOVA are displayed in Tables 2a and 2b. Activation of muscles during P0 was neither affected by RFD nor by IMP. This timeframe was considered the background activity (BGA). During P1 muscle activity revealed no significant changes when IMP was altered, while RFD significantly influenced activation of ST, BF and VM, but not of VL. Alteration of RFD did not significantly affect neuromuscular activation during P2 or P3, whereas enhancement of IMP was accomplished by significant increase in reflex activation during P2 and P3 in all muscles investigated.

The increase of neuromuscular activation was no longer significant during P3 for the knee flexors (ST, BF) when the reflex gain in relation to the BGA was considered instead of absolute values (Table 3).

5. Discussion

The results of our study showed relationships between stimulus characteristics of translational perturbations of the knee joint, namely IMP and RFD, and distinct parts of muscular responses of knee stabilizing muscles.

For P1 but not for P2, neuromuscular activation was dependent on RFD in both hamstring (ST and BF) and quadriceps (only VM) muscles. These findings correspond to the results of Grey and co-workers, who reported the SLR of the ankle muscle soleus being velocity sensitive while the MLR was not (Grey et al., 2001). These previous results, which were achieved during perturbation of gait, have been confirmed in isolated rotational perturbation of the ankle joint in an ergometer (Gollhofer & Rapp, 1993; Leukel et al., 2009). Reflex activation at the beginning of SLR has been attributed to fast conducting Ia fibres (Bove et al., 2003; Morin & Pierrot-Deseilligny, 1977).

In accordance to Diener and co-workers, who showed a significant correlation of the size of the MLR but not of the SLR in the ankle muscle triceps surae with the amplitude of perturbation, activation during P2 but not during P1 of the investigated knee joint muscles in the present study was modulated by IMP (Diener, Dichgans, Bootz, & Bacher, 1984). The same conclusions concerning the relationship between stretch amplitude and the size of MLR have been made when applying isolated rotational perturbations of the ankle joint (Gollhofer & Rapp, 1993; Leukel et al., 2009). Reflex activation during MLR was hypothesized to be mediated mainly by group II fibres with oligosynaptic connections to the spinal motoneurons in soleus muscle (Shoji, Kobayashi, Ushiba, Kagamihara, & Masakado, 2005) and flexor digitorum brevis muscle (Corna, Grasso, Nardone, & Schieppati, 1995).

In contrast to muscles acting at the ankle joint, responses of both the agonists as well as part of the antagonists (ST, BF and VM) in the present study were dependent on stimulus velocity during P1, whereas responses of all muscles during P2 were amplitude dependent. Hamstring muscles are acting synergistically to the ACL during anterior tibial translation whereas quadriceps muscles are hamstring and ACL antagonists. Consequently, in the present study a translational perturbation of the tibia in anterior direction resulted in a stretch of the hamstring muscles while the quadriceps was unloaded. This indicates that the activation of quadriceps and hamstring muscles might differ with respect to the involved afferent pathways. The following suggestions can be taken into account for afferents contributing to the responses and pathways involved.

5.1. Hamstring responses to the perturbation stimulus

In order to estimate the possible influence of joint rotation on muscle lengthening and subsequent reflex responses during the perturbation, preliminary investigations were performed. High speed video analysis suggested that the perturbation resulted in slight knee flexion, while the trunk remained in its position due to its mass inertia. Although hip joint rotation was not quantified, it is assumed that during the perturbation, the dynamical coupling of hip and knees resulted in hip extension with an amplitude and velocity comparable to that of the knee flexion. It is thereby suggested that the length of BF and ST was not dramatically altered during the perturbation by either knee or hip joint rotation.

Thus stretching of the hamstring muscles caused by the anterior tibial translation is assumed to be the major mechanical event that elicits hamstring reflex responses, whereas the influence of either knee or hip joint rotation seems to be of minor relevance for the activation of these muscles.

Friemert and co-workers provided reasonable evidence that muscular responses of the hamstring muscles to anterior tibial translation mainly originate from hamstring muscle primary and secondary spindle afferents (Friemert, Franke, Gollhofer, Claes, & Faist, 2010). While the design of the study allowed the authors to draw conclusions regarding afferent pathways of the homonymous muscle involved in the reflex response, a potential influence of heteronymous afferents on hamstring activation could not be excluded.

Afferents are known to influence agonist/antagonist muscle activation acting at the same joint as well as heteronymous muscle activation acting at the distal or proximal joint (Marchand-Pauvert, Nicolas, Marque, Iglesias, & Pierrot-Deseilligny, 2005; Marque, Nicolas, Simonetta-Moreau, Pierrot-Deseilligny, & Marchand-Pauvert, 2005; McClelland, Miller, & Eyre, 2001; Meunier, Penicaud, Pierrot-Deseilligny, & Rossi, 1990). Simonetta-Moreau, Marque, Marchand-Pauvert, and Pierrot-Deseilligny (1999) identified heteronymous facilitation from deep peroneal nerve to motoneurons of BF as well as from superficial peroneal nerve and gastrocnemius medialis nerve to ST motoneurons. A perturbation stimulus applied to the posterior aspect of the shank resulting in anterior tibial translation like in the present study could mechanically affect triceps surae muscles in a percussion like manner and thereby elicit afferents resulting in heteronymous activation of hamstring muscles either due to mechanical spread of the percussion stimulus or by radiation of the activity carried in triceps surae muscle afferents to the respective upper thigh muscles (Burke, Gandevia, & McKeon, 1983; O'Sullivan, Eyre, & Miller, 1991). Group Ia and group II afferents from triceps surae would be fast enough to influence hamstring as well as quadriceps activation during P1 as well as P2 (Roujeau, Decq, & Lefaucheur, 2004; Simonetta-Moreau et al., 1999).

5.2. Quadriceps responses to the perturbation stimulus

Interpretations regarding the afferent origin of quadriceps reflex responses to the perturbation are much more ambiguous than interpretations of hamstring responses, because quadriceps is not stretched by anterior tibial translation and homonymous Ia and II excitation of quadriceps is less likely.

Stretching of the extensors could have been the result of unloading the shank after the impulse has been transferred, but this should not occur until the tibia had started its translation back into the posterior direction. This is the case not earlier than the maximal tibial translation into anterior direction has been reached. Anterior tibial translation to its maximum lasted at least 48 ms after its onset (condition HIMP, Fig. 2). Assuming fast Ia afferents of the extensors being elicited by the posterior translation of the tibia on its way back, extensor responses with a latency of 20 ms should occur no earlier than 68 ms (48 + 20 ms) after the onset of the anterior tibial translation. Thus, posterior tibial translation and subsequent quadriceps lengthening due to unloading of the shank could have influenced activation during P3, but not during P1 or P2 (Fig. 2).

Mrachacz-Kersting and co-workers reported on quadriceps and hamstring reflex responses to sudden unexpected knee flexion perturbation during walking. Due to the fact that the responses of both muscle groups had almost identical onset latencies ranging from 23 ± 1 ms to 24 ± 3 ms, a functional excitatory connection between knee extensors and flexors was assumed (Mrachacz-Kersting et al., 2004). A significant delay of 5–8 ms of the onset of the quadriceps responses compared to the onset of hamstring responses in our study argues against a common monosynaptic Ia afferent pathway originating in the hamstring muscles and activating both flexors and extensors during anterior tibial translation.

In contrast, Ib afferents originating from Golgi tendon organs (GTO) of the stretched hamstring tendons could elicit antagonist activation of the quadriceps muscle during P1. As only few more synaptic transmissions are required and Ib afferents are travelling with almost the velocity of Ia afferents, this pathway should add approximately 2–5 ms to the travelling time of the homonymous hamstring Ia pathway and thus would have a similar latency as the neuromuscular responses we measured for quadriceps muscle (Pierrot-Deseilligny, Katz, & Morin, 1979). If there was a contribution of hamstring

Ib afferents, its influence on homonymous and on heteronymous muscles would not necessarily have to be of inhibitory nature (Dietz, Gollhofer, Kleiber, & Trippel, 1992; Faist et al., 2006; Stephens & Yang, 1996).

Alternatively to the contribution of heteronymous afferents originating from hamstring muscles, Ia and II afferents from other leg muscles could affect quadriceps activation. Simonetta-Moreau and co-workers found heteronymous facilitation transmitted via Ia and II afferents to motoneurons of the quadriceps not only from biceps femoris nerve, but from common peroneal nerve as well (Simonetta-Moreau et al., 1999). Thus afferents from both hamstring muscles as well as triceps surae muscles could have an excitatory influence on quadriceps activation. The influence of group I afferents from triceps surae muscles on quadriceps reflexes might itself be influenced by cutaneous afferents from ipsilateral and contralateral foot sole, so that alteration of ground contact during the perturbation in this study might have altered quadriceps responses (Bergego, Pierrot-Deseilligny, & Mazieres, 1981).

While VL did not display any relation to the stimulus characteristics during P1, VM responses during P1 were related to RFD. Thus, activation during the P1 timeframe may be attributed to heteronymous group I afferents at least for VM. The relation of IMP to quadriceps responses during P2 supports the assumption that heteronymous activation mediated via group II afferents might contribute to quadriceps activation during this timeframe.

Burke and co-workers found a long latency facilitation of quadriceps excitability by cutaneous afferents stimulated at the calf. This facilitation begun at a conditioning interval 60–150 ms prior to the quadriceps tendon tap (Burke, Kamen, & Koceja, 1989). Assuming that cutaneous afferents were elicited at the calf by the circular band-sling applied around the shank during the perturbation in this study, they might have even potentiated the afferents elicited by the quadriceps lengthening due to the unloading mechanism described above. Considering the time constraints, the contribution of cutaneous afferents should have occurred predominantly during P3. In addition to the mechanism described, postural responses and supraspinal contributions have to be considered relevant for quadriceps activation during P3 (Diener et al., 1984; Horak & Nashner, 1986; Mrachacz-Kersting, Grey, & Sinkjaer, 2006; Nashner, 1976) While IMP had significant influence on reflex size, RFD didn't systematically alter neuromuscular activation during P3.

Due to the fact that the perturbation stimulus was induced unilaterally, a pelvis rotation along the longitudinal axis of the body might have occurred. This movement could have elicited afferents from the contralateral side of the body which might have influenced quadriceps activation of the investigated leg. Using patellar tendon taps for the conditioning and the test stimulus, Kamen and Koceja found a short latency inhibition of ipsilateral quadriceps responses at 25 ms and a longer latency facilitation at 75 ms after the contralateral conditioning stimulus (Kamen & Koceja, 1989). Assuming that alteration of contralateral quadriceps length due to pelvis rotation starts with the ipsilateral anterior tibial displacement, the short latency inhibition should have influenced the onset of ipsilateral quadriceps responses during P1, while the longer latency facilitation should have been effective during P3.

Besides the heteronymous excitatory influences discussed above, potential inhibitory influences on quadriceps activation from various other structures should not be neglected. The activation of quadriceps might be affected by either reciprocal Ia inhibition from hamstring or by group Ib, II or III inhibition originating in different receptor systems located in knee joint structures (Freeman & Wyke, 1966; Grüber, Wolter, & Lierse, 1986; Raunest, Sager, & Bürgener, 1996). While Ib inhibition of quadriceps released by Golgi endings located in the ACL may be regarded relevant at the onset of activation, reciprocal Ia inhibition from hamstring to quadriceps should be depressed during functional tasks like joint stabilization (Bayoumi & Ashby, 1989; Bonnard, Camus, Coyle, & Pailhous, 2002; Nielsen & Kagamihara, 1992).

5.3. Functional considerations

Active joint stabilisation during perturbation relies on preactivation of the respective muscles and on reflex responses related to the stimulus (Dhaher et al., 2005). The selective responsiveness of distinct reflex components observed in our study provides the knee joint with protective mechanisms against various kinds of perturbations. As the fastest responses (P1) were sensitive to the velocity

of the stimulus (RFD), they are appropriate to decelerate fast perturbations in single events like sudden muscle lengthening during unexpected joint rotation or displacement (Friemert et al., 2010). The slower responses (P2) were related to the amplitude (IMP) of the stimulus and thereby fit well for the compensation of slower perturbations with ample extent, when a fast but brief burst would not be sufficient to counteract the anterior tibial translation due to the ongoing impact of the perturbation (Beard, Kyberd, O'connor, Fergusson, & Dodd, 1994; Jennings & Seedhom, 1994).

The observations made in our study can be implemented into prevention and rehabilitation training of ACL injuries where perturbations with anterior tibial translation and subsequent reflex activation are suggested (Fitzgerald, Axe, & Snyder-Mackler, 2000). Recently, Friemert and co-workers provided evidence that hamstring group I afferents may reduce maximal tibial translation in anteriorly directed perturbation of functional knee stability in healthy subjects (Friemert et al., 2010). If training for prevention of ACL injuries targets at this SLR mechanism, the perturbations that are applied during training do not necessarily require extensive amplitude but ample velocity. In case of ACL deficiency, a reduction of MLR has proven to be related to the giving way symptom (Melnyk, Faist, Gothner, Claes, & Friemert, 2007). Depending on the condition of the patient, perturbations with minor velocity and moderate amplitude targeting at MLR might be used for the rehabilitation training of ACL injury. Perturbations with pronounced amplitude and low velocity overcoming the mass inertia of the subject are associated with postural control rather than with joint stabilization, as the major part of reflex responses to this kind of perturbation are due during LLR (Diener et al., 1984; Horak & Nashner, 1986; Nashner, 1976).

Isolated hamstring activation is recommended for ACL rehabilitation as it can directly counteract anterior tibial translation and thereby reduces ACL strain (McNair, Wood, & Marshall, 1992; Tsuda, Okamura, Otsuka, Komatsu, & Tokuya, 2001). However, the role of quadriceps activation for functional knee joint stability should not be neglected in connection with ACL injury. During ski landing impact, quadriceps activation is supposed to be one of the key contributors to anterior tibial loading (Yeow, Lee, & Goh, 2010). Isolated quadriceps activation in non-weight bearing condition is able to enhance anterior tibial translation (Torzilli, Deng, & Warren, 1994; Yeow et al., 2010). It thereby acts antagonistically to the ACL and should be avoided during acute rehabilitation of injury (Kvist, 2005). When transitioning from non-weight bearing to weight bearing while walking, increasing axial loading is associated with increasing anterior tibial translation. During weight acceptance, hamstring and quadriceps muscles of the respective leg are therefore co-activated (Schmitz, Kim, & Shultz, 2010). As hamstring-quadriceps co-contraction contributes to functional knee stability by improving joint stiffness, prevention and rehabilitation of ACL injury should aim at this synergism (Baratta et al., 1988; Hirokawa, 1991; Solomonow & Krogsgaard, 2001). It could be shown in this study that during axial loading of the leg, sudden anterior tibial translation caused both hamstring and quadriceps responses. The activation of quadriceps started only slightly delayed compared to the activation of hamstring muscles resulting in the desired co-activation. A prevention training which contains perturbation exercises for the knee joint during axial loading increased reflex responses to anterior tibial displacement of hamstring as well as quadriceps muscles leading to intensified co-contraction and enhanced knee joint stiffness (Gruber, Bruhn, & Gollhofer, 2006).

Conflict of interest statement

The authors disclose any actual or potential conflict of interest that could inappropriately influence, or be perceived to influence, their work.

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References

Baratta, R., Solomonow, M., Zhou, B. H., Letson, D., Chuingard, R., & D'ambrosia, R. (1988). Muscular coactivation. The role of the antagonist musculature in maintaining knee stability. *American Journal of Sports Medicine, 2*, 113–122.

Bayoumi, A., & Ashby, P. (1989). Projections of group-Ia afferents to motoneurons of thigh muscles in man. *Experimental Brain Research, 76*, 223–228.
Beard, D. J., Kyberd, P. J., O'connor, J. J., Fergusson, C. M., & Dodd, C. A. (1994). Reflex hamstring contraction latency in anterior cruciate ligament deficiency. *Journal of Orthopaedic Research, 12*, 219–228.
Beck, I., Reichelt, A., & Beck, W. (2002). Klinische Ergebnisse der ambulanten Rehabilitaiton nach vorderer Kreuzbandplastik. *Arthroskopie, 15*, 17–20.
Bergego, C., Pierrot-Deseilligny, E., & Mazieres, L. (1981). Facilitation of transmission in Ib pathways by cutaneous afferents from the contralateral foot sole in man. *Neuroscience Letters, 27*, 297–301.
Bonnard, M., Camus, M., Coyle, T., & Pailhous, J. (2002). Task-induced modulation of motor evoked potentials in upper-leg muscles during human gait: A TMS study. *European Journal of Neuroscience, 16*, 2225–2230.
Bove, M., Nardone, A., & Schieppati, M. (2003). Effects of leg muscle tendon vibration on group Ia and group II reflex responses to stance perturbation in humans. *Journal of Physiology, 550*, 617–630.
Burke, D., Gandevia, S. C., & McKeon, B. (1983). The afferent volleys responsible for spinal proprioceptive reflexes in man. *Journal of Physiology (London), 339*, 535–552.
Burke, J. R., Kamen, G., & Koceja, D. M. (1989). Long-latency enhancement of quadriceps excitability from stimulation of skin afferents in young and old adults. *Journal of Gerontology, 44*, M158–M163.
Corna, S., Grasso, M., Nardone, A., & Schieppati, M. (1995). Selective depression of medium-latency leg and foot muscle responses to stretch by an alpha 2-agonist in humans. *Journal of Physiology, 484*(Pt 3), 803–809.
De Luca, C. J. (1997). The use of surface electromyography in biomechanics. *Journal of Applied Biomechanics, 13*, 135–163.
Dhaher, Y. Y., Tsoumanis, A. D., Houle, T. T., & Rymer, W. Z. (2005). Neuromuscular reflexes contribute to knee stiffness during valgus loading. *Journal of Neurophysiology, 93*, 2698–2709.
Diener, H. C., Dichgans, J., Bootz, F., & Bacher, M. (1984). Early stabilization of human posture after a sudden disturbance: Influence of rate and amplitude of displacement. *Experimental Brain Research, 56*, 126–134.
Dietz, V., & Duysens, J. (2000). Significance of load receptor input during locomotion: A review. *Gait Posture, 11*, 102–110.
Dietz, V., Gollhofer, A., Kleiber, M., & Trippel, M. (1992). Regulation of bipedal stance: Dependency on load receptors. *Experimental Brain Research, 89*, 229–231.
Faist, M., Hoefer, C., Hodapp, M., Dietz, V., Berger, W., & Duysens, J. (2006). In humans Ib facilitation depends on locomotion while suppression of Ib inhibition requires loading. *Brain Research, 1076*, 87–92.
Fitzgerald, G. K., Axe, M. J., & Snyder-Mackler, L. (2000). The efficacy of perturbation training in nonoperative anterior cruciate ligament rehabilitation programs for physical active individuals. *Physical Therapy, 80*, 128–140.
Freeman, M. A., & Wyke, B. (1966). Articular contributions to limb muscle reflexes. The effects of partial neurectomy of the knee-joint on postural reflexes. *British Journal of Surgery, 53*, 61–68.
Friemert, B., Franke, S., Gollhofer, A., Claes, L., & Faist, M. (2010). Group I afferent pathway contributes to functional knee stability. *Journal of Neurophysiology, 103*, 616–622.
Gollhofer, A., & Rapp, W. (1993). Recovery of stretch reflex responses following mechanical stimulation. *European Journal of Applied Physiology and Occupational Physiology, 66*, 415–420.
Grey, M. J., Ladouceur, M., Andersen, J. B., Nielsen, J. B., & Sinkjaer, T. (2001). Group II muscle afferents probably contribute to the medium latency soleus stretch reflex during walking in humans. *Journal of Physiology, 534*, 925–933.
Gruber, M., Bruhn, S., & Gollhofer, A. (2006). Specific adaptations of neuromuscular control and knee joint stiffness following sensorimotor training. *International Journal of Sports Medicine, 27*, 636–641.
Grüber, J., Wolter, D., & Lierse, W. (1986). Der vordere Kreuzbandreflex (LCA-Reflex). *Unfallchirurg, 89*, 551–554.
Hirokawa, S. (1991). Muscular co-contraction and control of knee stability. *Journal of Electromyography and Kinesiology, 1*, 199–208.
Horak, F., & Nashner, L. (1986). Central programming of postural movements: Adaptation to altered support-surface configurations. *Journal of Neurophysiology, 55*, 1369–1381.
Jennings, A. G., & Seedhom, B. B. (1994). Proprioception in the knee and reflex hamstring contraction latency. *Journal of Bone and Joint Surgery [Br], 3*, 491–494.
Kamen, G., & Koceja, D. M. (1989). Contralateral influences on patellar tendon reflexes in young and old adults. *Neurobiology and Aging, 10*, 311–315.
Kvist, J. (2005). Sagittal tibial translation during exercises in the anterior cruciate ligament-deficient knee. *Scandinavian Journal of Medicine and Science in Sports, 15*, 148–158.
Leukel, C., Lundbye-Jensen, J., Gruber, M., Zuur, A., Gollhofer, A., & Taube, W. (2009). Short-term pressure induced suppression of the short-latency response: A new methodology for investigating stretch reflexes. *Journal of Applied Physiology, 107*, 1051–1058.
Marchand-Pauvert, V., Nicolas, G., Marque, P., Iglesias, C., & Pierrot-Deseilligny, E. (2005). Increase in group II excitation from ankle muscles to thigh motoneurones during human standing. *Journal of Physiology, 566*, 257–271.
Markolf, K. L., Graff-Radford, A., & Amstutz, H. C. (1978). In vivo knee stability. A quantitative assessment using an instrumented clinical testing apparatus. *Journal of Bone and Joint Surgery, 5*, 664–674.
Marque, P., Nicolas, G., Simonetta-Moreau, M., Pierrot-Deseilligny, E., & Marchand-Pauvert, V. (2005). Group II excitations from plantar foot muscles to human leg and thigh motoneurones. *Experimental Brain Research, 161*, 486–501.
McClelland, V., Miller, S., & Eyre, J. (2001). Short latency heteronymous excitatory and inhibitory reflexes between antagonist and heteronymous muscles of the human shoulder and upper limb. *Brain Research, 899*, 82–93.
McNair, P. J., Wood, G. A., & Marshall, R. N. (1992). Stiffness of the hamstring muscles and its relationship to function in anterior cruciate ligament deficient individuals. *Clinical Biomechanics, 7*, 131–137.
Melnyk, M., Faist, M., Gothner, M., Claes, L., & Friemert, B. (2007). Changes in stretch reflex excitability are related to ''giving way'' symptoms in patients with anterior cruciate ligament rupture. *Journal of Neurophysiology, 97*, 474–480.
Meunier, S., Penicaud, A., Pierrot-Deseilligny, E., & Rossi, A. (1990). Monosynaptic Ia excitation and recurrent inhibition from quadriceps to ankle flexors and extensors in man. *Journal of Physiology, 423*, 661–675.
Morin, C., & Pierrot-Deseilligny, E. (1977). Role of Ia afferents in the soleus motoneurones. Inhibition during a tibialis anterior voluntary contraction in man. *Experimental Brain Research, 27*, 509–522.

Mrachacz-Kersting, N., Grey, M. J., & Sinkjaer, T. (2006). Evidence for a supraspinal contribution to the human quadriceps long-latency stretch reflex. *Experimental Brain Research, 168*, 529–540.
Mrachacz-Kersting, N., Lavoie, B. A., Andersen, J. B., & Sinkjaer, T. (2004). Characterisation of the quadriceps stretch reflex during the transition from swing to stance phase of human walking. *Experimental Brain Research, 159*, 108–122.
Nardone, A., & Schieppati, M. (1998). Medium-latency response to muscle stretch in human lower limb: Estimation of conduction velocity of group II fibres and central delay. *Neuroscience Letters, 249*, 29–32.
Nardone, A., & Schieppati, M. (2004). Group II spindle fibres and afferent control of stance. Clues from diabetic neuropathy. *Clinical Neurophysiology, 115*, 779–789.
Nashner, L. M. (1976). Adapting reflexes controlling the human posture. *Experimental Brain Research, 26*, 59–72.
Nebelung, W. (Ed.). (2006). *Kreuzbandverletzungen*. Berlin: Springer.
Nielsen, J., & Kagamihara, Y. (1992). The regulation of disynaptic reciprocal Ia inhibition during co-contraction of antagonistic muscles in man. *Journal of Physiology, 456*, 373–391.
O'connor, J. J. (1993). Can muscle co-contraction protect knee ligaments after injury or repair? *Journal of Bone and Joint Surgery [Br], 1*, 41–48.
Olmstead, T. G., Wevers, H. W., Bryant, J. T., & Gouw, G. J. (1986). Effect of muscular activity on valgus/varus laxity and stiffness of the knee. *Journal of Biomechanics, 19*, 565–577.
O'Sullivan, M., Eyre, J., & Miller, S. (1991). Radiation of phasic stretch reflex in biceps brachii to muscles of the arm in man and its restriction during development. *Journal of Physiology, 439*, 529–543.
Pierrot-Deseilligny, E., Katz, R., & Morin, C. (1979). Evidence for Ib inhibition in human subjects. *Brain Research, 166*, 176–179.
Raunest, J., Sager, M., & Bürgener, E. (1996). Proprioceptive mechanisms in the cruciate ligaments: An electromyographic study on reflex activity in the thigh muscles. *Journal of Trauma, 41*, 488–493.
Roujeau, T., Decq, P., & Lefaucheur, J. (2004). Surface EMG recording of heteronymous reflex excitation of semitendinosus motoneurones by group II afferents. *Clinical Neurophysiology, 115*, 1313–1319.
Schabus, R., & Bosina, E. (2007). *Das Knie*. Wien: Springer.
Schmitz, R., Kim, H., & Shultz, S. (2010). Effect of axial load on anterior tibial translation when transitioning from non-weight bearing to weight bearing. *Clinical Biomechanics (Bristol, Avon), 25*, 77–82.
Shoji, J., Kobayashi, K., Ushiba, J., Kagamihara, Y., & Masakado, Y. (2005). Inhibition from the plantar nerve to soleus muscle during the stance phase of walking. *Brain Research, 1048*, 48–58.
Simonetta-Moreau, M., Marque, P., Marchand-Pauvert, V., & Pierrot-Deseilligny, E. (1999). The pattern of excitation of human lower limb motoneurones by probable group II muscle afferents. *Journal of Physiology, 517*, 287–300.
Solomonow, M., & Krogsgaard, M. (2001). Sensorimotor control of knee stability: A review. *Scandinavian Journal of Medicine and Science and Sports, 11*, 64–80.
Stephens, M. J., & Yang, J. F. (1996). Short latency, non-reciprocal group I inhibition is reduced during the stance phase of walking in humans. *Brain Research, 743*, 24–31.
Torzilli, P. A., Deng, X., & Warren, R. F. (1994). The effect of joint-compressive load and quadriceps muscle force on knee motion in the intact and anterior cruciate ligament-sectioned knee. *American Journal of Sports Medicine, 1*, 105–112.
Tsuda, E., Okamura, Y., Otsuka, H., Komatsu, T., & Tokuya, S. (2001). Direct evidence of the anterior cruciate ligament-hamstring reflex arc in humans. *American Journal of Sports Medicine, 29*, 83–87.
Wojtys, E. M., & Huston, L. J. (1994). Neuromuscular performance in normal and anterior cruciate ligament-deficient lower extremities. *American Journal of Sports Medicine, 22*, 89–104.
Yeow, C., Lee, P., & Goh, J. (2010). Direct contribution of axial impact compressive load to anterior tibial load during simulated ski landing impact. *Journal of Biomechanics, 43*, 242–247.